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NEWS	1			Web Page URLs for STN Seminar Schedule - N. America
NEWS	2			"Ask CAS" for self-help around the clock
NEWS	3	FEB	25	CA/CAPLUS - Russian Agency for Patents and Trademarks (ROSPATENT) added to list of core patent offices covered
NEWS	4	FEB	28	PATDPAFULL - New display fields provide for legal status data from INPADOC
NEWS	5	FEB	28	BABS - Current-awareness alerts (SDIs) available
NEWS	6	FEB	28	MEDLINE/LMEDLINE reloaded
NEWS	7	MAR	02	GBFULL: New full-text patent database on STN
NEWS	8	MAR	03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	9	MAR	03	MEDLINE file segment of TOXCENTER reloaded
NEWS	10	MAR	22	KOREAPAT now updated monthly; patent information enhanced
NEWS	11	MAR	22	Original IDE display format returns to REGISTRY/ZREGISTRY
NEWS	12	MAR	22	PATDPASPC - New patent database available
NEWS	13	MAR	22	REGISTRY/ZREGISTRY enhanced with experimental property tags
NEWS	14	APR	04	EPFULL enhanced with additional patent information and new fields
NEWS	15	APR	04	EMBASE - Database reloaded and enhanced
NEWS	16	APR	18	New CAS Information Use Policies available online
NEWS	17	APR	25	Patent searching, including current-awareness alerts (SDIs), based on application date in CA/CAPLUS and USPATFULL/USPAT2 may be affected by a change in filing date for U.S. applications.
NEWS	18	APR	28	Improved searching of U.S. Patent Classifications for U.S. patent records in CA/CAPLUS
NEWS	EXPRESS			JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005
NEWS	HOURS			STN Operating Hours Plus Help Desk Availability
NEWS	INTER			General Internet Information
NEWS	LOGIN			Welcome Banner and News Items
NEWS	PHONE			Direct Dial and Telecommunication Network Access to STN
NEWS	WWW			CAS World Wide Web Site (general information)

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\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 17:29:33 ON 17 MAY 2005

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=> file medline, uspatful, dgene, embase, wpids, fsta, jicst, biosis, biobusiness, ceaba
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
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FILE 'MEDLINE' ENTERED AT 17:30:02 ON 17 MAY 2005

FILE 'USPATFULL' ENTERED AT 17:30:02 ON 17 MAY 2005  
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FILE 'CEABA-VTB' ENTERED AT 17:30:02 ON 17 MAY 2005  
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=> s smurf and smad  
L1 25 SMURF AND SMAD

=> s PPXY domain and Smurf WW domain  
L2 1 PPXY DOMAIN AND SMURF WW DOMAIN

=> d l2 ti abs ibib tot

L2 ANSWER 1 OF 1 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN  
TI Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.  
AN 2001-071267 [08] WPIDS  
AB WO 200077168 A UPAB: 20011129  
NOVELTY - An isolated Smurf1 or Smurf2 protein (I), is new.  
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:  
(1) an isolated nucleic acid (II) encoding (I);  
(2) a vector (III) comprising (II);  
(3) a host cell (IV) comprising (III);  
(4) production of (I);  
(5) a transgenic non-human animal that expresses a human (I);  
(6) screening (M) for a modulator of Smurf activity, comprising detecting modulation of Smurf activity in the presence of a test compound relative to Smurf activity in the absence of the test compound;  
(7) an antibody (V) that specifically binds to (I);  
(8) an oligonucleotide or nucleic acid (VI) that specifically hybridizes to (II) under highly stringent conditions; and  
(9) promoting a bone morphogenic protein or transforming growth factor (TGF)- beta activation pathway in a cell, comprising suppressing expression of endogenous Smurf in the cell.  
ACTIVITY - Cytostatic.  
MECHANISM OF ACTION - Negative regulator of Smad signal transduction; antagonist of BMP and TGF- beta signaling pathway.  
The inhibition of Smad1 by Smurf1 was tested. By over expressing Smad1 and Smad2 together with various dosages of Smurf1 in Xenopus animal caps, the ability of Smurf1 to directly antagonize the mesoderm induction

activities of Smad1 and Smad2, was tested. The results showed that expression of Smad1 alone induced ventral mesoderm, as demonstrated by expression of the ventral/posterior mesodermal markers Xhox3 and Xcad1. However, co-expression of Smurf1 and Smad1 blocked induction of these markers at all Smurf1 doses tested, demonstrating that Smurf1 can antagonize Smad1 activity.

USE - Expression of (I) from (III) in a cell is useful for inhibiting a bone morphogenic protein (BMP) or transforming growth factor- beta (TGF beta ) activation pathway in a cell (claimed). (I) is useful to block chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. (I) is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it by antagonizing or mimicking the activity of (I), respectively, and in screening assays for identifying specific ligands of (I). (I) is useful as an immunogen to generate antibodies that are useful to alter the BMP pathway by inhibiting (I) or for diagnostic purposes. (I) is useful for treating a disorder associated with BMP or TGF- beta activation, such as cancer. (I) or inhibitor of (I) can be delivered by a vector to modulate Smads, e.g. to prevent Smurf regulation of Smads where BMP or TGF beta activity is desired, such as in bone regeneration or to study Smurf regulator processes in vivo.

Dwg.0/18

ACCESSION NUMBER: 2001-071267 [08] WPIDS  
 DOC. NO. CPI: C2001-019969  
 TITLE: Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.  
 DERWENT CLASS: B04 D16  
 INVENTOR(S): THOMSEN, G H; WRANA, J  
 PATENT ASSIGNEE(S): (HSCR-N) HSC RES & DEV LP; (UYN Y) UNIV NEW YORK STATE RES FOUND  
 COUNTRY COUNT: 93  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000077168	A2	20001221	(200108)*	EN	106
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000056107	A	20010102	(200121)		
EP 1192174	A2	20020403	(200230)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2003502064	W	20030121	(200308)		131
CN 1409722	A	20030409	(200345)		

#### APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000077168	A2	WO 2000-US16250	20000612
AU 2000056107	A	AU 2000-56107	20000612
EP 1192174	A2	EP 2000-941398	20000612
		WO 2000-US16250	20000612
JP 2003502064	W	WO 2000-US16250	20000612
		JP 2001-504003	20000612
CN 1409722	A	CN 2000-811354	20000612

#### FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000056107	A Based on	WO 2000077168
EP 1192174	A2 Based on	WO 2000077168
JP 2003502064	W Based on	WO 2000077168

PRIORITY APPLN. INFO: US 1999-138969P 19990611

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(FILE 'HOME' ENTERED AT 17:29:33 ON 17 MAY 2005)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS, BIOSIS, BIOBUSINESS, CEABA-VTB' ENTERED AT 17:30:02 ON 17 MAY 2005

L1 25 S SMURF AND SMAD  
L2 1 S PPXY DOMAIN AND SMURF WW DOMAIN

=> d ll ti abs ibib tot

L1 ANSWER 1 OF 25 MEDLINE on STN  
TI NEDD4-2 (neural precursor cell expressed, developmentally down-regulated 4-2) negatively regulates TGF-beta (transforming growth factor-beta) signalling by inducing ubiquitin-mediated degradation of Smad2 and TGF-beta type I receptor.  
AB Inhibitory **Smad**, Smad7, is a potent inhibitor of TGF-beta (transforming growth factor-beta) superfamily signalling. By binding to activated type I receptors, it prevents the activation of R-Smads (receptor-regulated Smads). To identify new components of the **Smad** pathway, we performed yeast two-hybrid screening using Smad7 as bait, and identified NEDD4-2 (neural precursor cell expressed, developmentally down-regulated 4-2) as a direct binding partner of Smad7. NEDD4-2 is structurally similar to Smurfs (**Smad** ubiquitin regulatory factors) 1 and 2, which were identified previously as E3 ubiquitin ligases for R-Smads and TGF-beta superfamily receptors. NEDD4-2 functions like Smurfs 1 and 2 in that it associates with TGF-beta type I receptor via Smad7, and induces its ubiquitin-dependent degradation. Moreover, NEDD4-2 bound to TGF-beta-specific R-Smads, Smads 2 and 3, in a ligand-dependent manner, and induced degradation of Smad2, but not Smad3. However, in contrast with Smurf2, NEDD4-2 failed to induce ubiquitination of SnoN (Ski-related novel protein N), although NEDD4-2 bound to SnoN via Smad2 more strongly than Smurf2. We showed further that overexpressed NEDD4-2 prevents transcriptional activity induced by TGF-beta and BMP, whereas silencing of the NEDD4-2 gene by siRNA (small interfering RNA) resulted in enhancement of the responsiveness to TGF-beta superfamily cytokines. These data suggest that NEDD4-2 is a member of the **Smurf**-like C2-WW-HECT (WW is Trp-Trp and HECT is homologous to the E6-accessory protein) type E3 ubiquitin ligases, which negatively regulate TGF-beta superfamily signalling through similar, but not identical, mechanisms to those used by Smurfs.

ACCESSION NUMBER: 2005112864 IN-PROCESS  
DOCUMENT NUMBER: PubMed ID: 15496141  
TITLE: NEDD4-2 (neural precursor cell expressed, developmentally down-regulated 4-2) negatively regulates TGF-beta (transforming growth factor-beta) signalling by inducing ubiquitin-mediated degradation of Smad2 and TGF-beta type I receptor.  
AUTHOR: Kuratomi Go; Komuro Akiyoshi; Goto Kouichiro; Shinozaki Masahiko; Miyazawa Keiji; Miyazono Kohei; Imamura Takeshi  
CORPORATE SOURCE: Department of Biochemistry, The Cancer Institute of the Japanese Foundation for Cancer Research (JFCR), 1-37-1 Kami-ikebukuro, Toshima-ku, Tokyo 170-8455, Japan.  
SOURCE: Biochemical journal, (2005 Mar 15) 386 (Pt 3) 461-70.  
Journal code: 2984726R. ISSN: 1470-8728.  
PUB. COUNTRY: England: United Kingdom  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English

FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals  
ENTRY DATE: Entered STN: 20050304  
Last Updated on STN: 20050309

L1 ANSWER 2 OF 25 MEDLINE on STN  
TI Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.  
AB **Smad** ubiquitin regulatory factor (**Smurf**) 1 binds to receptor-regulated Smads for bone morphogenetic proteins (BMPs) Smad1/5 and promotes their degradation. In addition, Smurf1 associates with transforming growth factor-beta type I receptor through the inhibitory **Smad** (I-**Smad**) Smad7 and induces their degradation. Herein, we examined whether Smurf1 negatively regulates BMP signaling together with the I-Smads Smad6/7. Smurf1 and Smad6 cooperatively induced secondary axes in *Xenopus* embryos. Using a BMP-responsive promoter-reporter construct in mammalian cells, we found that Smurf1 cooperated with I-**Smad** in inhibiting BMP signaling and that the inhibitory activity of Smurf1 was not necessarily correlated with its ability to bind to Smad1/5 directly. Smurf1 bound to BMP type I receptors via I-Smads and induced ubiquitination and degradation of these receptors. Moreover, Smurf1 associated with Smad1/5 indirectly through I-Smads and induced their ubiquitination and degradation. Smurf1 thus controls BMP signaling with and without I-Smads through multiple mechanisms.  
ACCESSION NUMBER: 2003328281 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 12857866  
TITLE: Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.  
AUTHOR: Murakami Gyo; Watabe Tetsuro; Takaoka Kunio; Miyazono Kohei; Imamura Takeshi  
CORPORATE SOURCE: Department of Biochemistry, The Cancer Institute of the Japanese Foundation for Cancer Research, Tokyo 170-8455, Japan.  
SOURCE: Molecular biology of the cell, (2003 Jul) 14 (7) 2809-17.  
Electronic Publication: 2003-04-04.  
Journal code: 9201390. ISSN: 1059-1524.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200404  
ENTRY DATE: Entered STN: 20030715  
Last Updated on STN: 20040414  
Entered Medline: 20040413

L1 ANSWER 3 OF 25 MEDLINE on STN  
TI A new **Smurf** in the village.  
AB TGF-beta signaling is modulated by Smurfs, E3-ubiquitin ligases that selectively target the receptors and **Smad** proteins for degradation. New evidence from *Drosophila* suggests that Smurfs regulate the amplitude and the duration of the cellular response to signaling in vivo.  
ACCESSION NUMBER: 2001654258 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 11703932  
TITLE: A new **Smurf** in the village.  
AUTHOR: Arora K; Warrior R  
CORPORATE SOURCE: Department of Developmental and Cell Biology, University of California, Irvine 92697, USA.  
SOURCE: Developmental cell, (2001 Oct) 1 (4) 441-2.  
Journal code: 101120028. ISSN: 1534-5807.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200112  
ENTRY DATE: Entered STN: 20011115  
Last Updated on STN: 20020123  
Entered Medline: 20011207

L1 ANSWER 4 OF 25 USPATFULL on STN  
TI Light probe for ultraviolet light activated gene transduction  
AB In accordance with the present invention, a light probe is provided for treating a patient through the use of ultraviolet light activated gene therapy. Embodiments of the present invention include a light probe structure for the utilization of light activated gene therapy to repair and/or rebuild damaged cartilage or a component of a functional spinal unit (FSU) by introducing a desired gene into a patient's tissue.

ACCESSION NUMBER: 2004:333777 USPATFULL  
TITLE: Light probe for ultraviolet light activated gene transduction  
INVENTOR(S): Schwarz, Edward M., Rochester, NY, UNITED STATES  
Rubery, Paul T., Honeoye Falls, NY, UNITED STATES  
Foster, Thomas H., Rochester, NY, UNITED STATES  
Maloney, Michael D., Pittsford, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004264853	A1	20041230
APPLICATION INFO.:	US 2004-769392	A1	20040130 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-444493P	20030131 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614	
NUMBER OF CLAIMS:	55	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	10 Drawing Page(s)	
LINE COUNT:	883	

L1 ANSWER 5 OF 25 USPATFULL on STN  
TI Light activated gene transduction using long wavelength ultraviolet light for cell targeted gene delivery  
AB In accordance with the present invention, methods are provided for treating a patient through the use of ultraviolet light activated gene therapy. Embodiments of the present invention include methods for the utilization of light activated gene therapy to repair and/or rebuild damaged cartilage by introducing a desired gene into a patient's tissue.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:335516 USPATFULL  
TITLE: Light activated gene transduction using long wavelength ultraviolet light for cell targeted gene delivery  
INVENTOR(S): Schwarz, Edward M., Rochester, NY, UNITED STATES  
O'Keefe, Regis J., Pittsford, NY, UNITED STATES  
Foster, Thomas, Rochester, NY, UNITED STATES  
Finlay, Jarod C., Philadelphia, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003236394	A1	20031225
APPLICATION INFO.:	US 2003-357271	A1	20030131 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-353842P	20020131 (60)
	US 2002-353907P	20020131 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614	
NUMBER OF CLAIMS:	75	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	9 Drawing Page(s)	

L1 ANSWER 6 OF 25 DGENE COPYRIGHT 2005 The Thomson Corp on STN  
TI Novel isolated **Smurf** protein useful for inhibiting bone  
morphogenic protein or tumor growth factor-beta activation pathway, for  
treating cancer and to block osteogenesis, hair growth, tooth formation  
-

AN AAB31477 Protein DGENE

AB The present sequence represents a human Smurf2 polypeptide. The  
specification also describes a Smurf1 polypeptide. **Smurf**  
polypeptides are negative regulators of **Smad** signal  
transduction, and antagonists of bone morphogenic protein (BMP) or  
transforming growth factor-beta (TGF-beta) signalling pathway. Expression  
of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation  
pathway in a cell. **Smurf** polypeptides are useful for blocking  
chondrogenesis, osteogenesis, blood differentiation, cartilage formation,  
neural tube patterning, retinal development, heart induction and  
morphogenesis, hair growth, tooth formation, gamete formation and a wide  
variety of tissue and organ formation processes, and hinder the  
regeneration, growth, maintenance, etc., of bone and other tissues that  
are dependent on the BMP pathway. The polypeptide is useful for screening  
for various drugs and/or antibodies that can either enhance the BMP  
pathway, or inhibit it.

ACCESSION NUMBER: AAB31477 Protein DGENE

TITLE: Novel isolated **Smurf** protein useful for inhibiting  
bone morphogenic protein or tumor growth factor-beta  
activation pathway, for treating cancer and to block  
osteogenesis, hair growth, tooth formation -

INVENTOR: Thomsen G H; Wrana J

PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.  
(HSCR-N) HSC RES & DEV LP.

PATENT INFO: WO 2000077168 A2 20001221 107

APPLICATION INFO: WO 2000-US16250 20000612

PRIORITY INFO: US 1999-138969 19990611

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: 2001-071267 [08]

CROSS REFERENCES: N-PSDB: AAF24853

DESCRIPTION: Amino acid sequence of a human Smurf2 polypeptide.

L1 ANSWER 7 OF 25 DGENE COPYRIGHT 2005 The Thomson Corp on STN  
TI Novel isolated **Smurf** protein useful for inhibiting bone  
morphogenic protein or tumor growth factor-beta activation pathway, for  
treating cancer and to block osteogenesis, hair growth, tooth formation  
-

AN AAB31476 Protein DGENE

AB The present sequence represents a human Smurf1 polypeptide. The  
specification also describes a Smurf2 polypeptide. **Smurf**  
polypeptides are negative regulators of **Smad** signal  
transduction, and antagonists of bone morphogenic protein (BMP) or  
transforming growth factor-beta (TGF-beta) signalling pathway. Expression  
of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation  
pathway in a cell. **Smurf** polypeptides are useful for blocking  
chondrogenesis, osteogenesis, blood differentiation, cartilage formation,  
neural tube patterning, retinal development, heart induction and  
morphogenesis, hair growth, tooth formation, gamete formation and a wide  
variety of tissue and organ formation processes, and hinder the  
regeneration, growth, maintenance, etc., of bone and other tissues that  
are dependent on the BMP pathway. The polypeptide is useful for screening  
for various drugs and/or antibodies that can either enhance the BMP  
pathway, or inhibit it.

ACCESSION NUMBER: AAB31476 Protein DGENE

TITLE: Novel isolated **Smurf** protein useful for inhibiting  
bone morphogenic protein or tumor growth factor-beta  
activation pathway, for treating cancer and to block  
osteogenesis, hair growth, tooth formation -

INVENTOR: Thomsen G H; Wrana J

PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.  
(HSCR-N) HSC RES & DEV LP.  
PATENT INFO: WO 2000077168 A2 20001221 107  
APPLICATION INFO: WO 2000-US16250 20000612  
PRIORITY INFO: US 1999-138969 19990611  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 2001-071267 [08]  
CROSS REFERENCES: N-PSDB: AAF24852  
DESCRIPTION: Amino acid sequence of a human Smurf1 polypeptide.

L1 ANSWER 8 OF 25 DGENE COPYRIGHT 2005 The Thomson Corp on STN  
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation

AN AAF24855 DNA DGENE  
AB PCR primers AAF24854-55 were used to amplify human Smurf2 cDNA. The specification also describes a Smurf1 polypeptide. **Smurf** polypeptides are negative regulators of **Smad** signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.

ACCESSION NUMBER: AAF24855 DNA DGENE  
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -

INVENTOR: Thomsen G H; Wrana J  
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.  
(HSCR-N) HSC RES & DEV LP.

PATENT INFO: WO 2000077168 A2 20001221 107  
APPLICATION INFO: WO 2000-US16250 20000612  
PRIORITY INFO: US 1999-138969 19990611  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 2001-071267 [08]  
DESCRIPTION: PCR primer used to amplify human Smurf1 cDNA.

L1 ANSWER 9 OF 25 DGENE COPYRIGHT 2005 The Thomson Corp on STN  
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation

AN AAF24854 DNA DGENE  
AB PCR primers AAF24854-55 were used to amplify human Smurf2 cDNA. The specification also describes a Smurf1 polypeptide. **Smurf** polypeptides are negative regulators of **Smad** signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP

pathway, or inhibit it.

ACCESSION NUMBER: AAF24854 DNA DGENE  
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -  
INVENTOR: Thomsen G H; Wrana J  
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.  
(HSCR-N) HSC RES & DEV LP.  
PATENT INFO: WO 2000077168 A2 20001221 107  
APPLICATION INFO: WO 2000-US16250 20000612  
PRIORITY INFO: US 1999-138969 19990611  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 2001-071267 [08]  
DESCRIPTION: PCR primer used to amplify human Smurf1 cDNA.

L1 ANSWER 10 OF 25 DGENE COPYRIGHT 2005 The Thomson Corp on STN  
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -

AN AAF24853 cDNA DGENE  
AB The present sequence encodes a human Smurf2 polypeptide. The specification also describes a Smurf1 polypeptide. **Smurf** polypeptides are negative regulators of **Smad** signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.

ACCESSION NUMBER: AAF24853 cDNA DGENE  
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -  
INVENTOR: Thomsen G H; Wrana J  
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.  
(HSCR-N) HSC RES & DEV LP.  
PATENT INFO: WO 2000077168 A2 20001221 107  
APPLICATION INFO: WO 2000-US16250 20000612  
PRIORITY INFO: US 1999-138969 19990611  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 2001-071267 [08]  
CROSS REFERENCES: P-PSDB: AAB31477  
DESCRIPTION: Nucleotide sequence of a human Smurf2 polypeptide.

L1 ANSWER 11 OF 25 DGENE COPYRIGHT 2005 The Thomson Corp on STN  
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -

AN AAF24852 cDNA DGENE  
AB The present sequence encodes a human Smurf1 polypeptide. The specification also describes a Smurf2 polypeptide. **Smurf** polypeptides are negative regulators of **Smad** signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking

chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.

ACCESSION NUMBER: AAF24852 cDNA DGENE  
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -  
INVENTOR: Thomsen G H; Wrana J  
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.  
(HSCR-N) HSC RES & DEV LP.  
PATENT INFO: WO 2000077168 A2 20001221 107  
APPLICATION INFO: WO 2000-US16250 20000612  
PRIORITY INFO: US 1999-138969 19990611  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 2001-071267 [08]  
CROSS REFERENCES: P-PSDB: AAB31476  
DESCRIPTION: Nucleotide sequence of a human Smurf1 polypeptide.

L1 ANSWER 12 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN

TI NEDD4-2 (neural precursor cell expressed, developmentally down-regulated 4-2) negatively regulates TGF- $\beta$  (transforming growth factor- $\beta$ ) signalling by inducing ubiquitin-mediated degradation of Smad2 and TGF- $\beta$  type I receptor.

AB Inhibitory **Smad**, Smad7, is a potent inhibitor of TGF- $\beta$  (transforming growth factor- $\beta$ ) superfamily signalling. By binding to activated type I receptors, it prevents the activation of R-Smads (receptor-regulated Smads). To identify new components of the **Smad** pathway, we performed yeast two-hybrid screening using Smad7 as bait, and identified NEDD4-2 (neural precursor cell expressed, developmentally down-regulated 4-2) as a direct binding partner of Smad7. NEDD4-2 is structurally similar to Smurfs (**Smad** ubiquitin regulatory factors) 1 and 2, which were identified previously as E3 ubiquitin ligases for R-Smads and TGF- $\beta$  super-family receptors. NEDD4-2 functions like Smurfs 1 and 2 in that it associates with TGF- $\beta$  type I receptor via Smad7, and induces its ubiquitin-dependent degradation. Moreover, NEDD4-2 bound to TGF- $\beta$ -specific R-Smads, Smads 2 and 3, in a ligand-dependent manner, and induced degradation of Smad2, but not Smad3. However, in contrast with Smurf2, NEDD4-2 failed to induce ubiquitination of SnoN (Ski-related novel protein N), although NEDD4-2 bound to SnoN via Smad2 more strongly than Smurf2. We showed further that overexpressed NEDD4-2 prevents transcriptional activity induced by TGF- $\beta$  and BMP, whereas silencing of the NEDD4-2 gene by siRNA (small interfering RNA) resulted in enhancement of the responsiveness to TGF- $\beta$  superfamily cytokines. These data suggest that NEDD4-2 is a member of the **Smurf**-like C2-WW-HECT (WW is Trp-Trp and HECT is homologous to the E6-accessory protein) type E3 ubiquitin ligases, which negatively regulate TGF- $\beta$  superfamily signalling through similar, but not identical, mechanisms to those used by Smurfs. .COPYRG. 2005 Biochemical Society.

ACCESSION NUMBER: 2005147324 EMBASE  
TITLE: NEDD4-2 (neural precursor cell expressed, developmentally down-regulated 4-2) negatively regulates TGF- $\beta$  (transforming growth factor- $\beta$ ) signalling by inducing ubiquitin-mediated degradation of Smad2 and TGF- $\beta$  type I receptor.  
AUTHOR: Kuratomi G.; Komuro A.; Goto K.; Shinozaki M.; Miyazawa K.; Miyazono K.; Imamura T.  
CORPORATE SOURCE: K. Miyazono, Department of Biochemistry, Cancer Institute, Japanese Found. for Cancer Research, 1-37-1 Kami-ikebukuro, Toshima-ku, Tokyo 170-8455, Japan. miyazono-ind@umin.ac.jp

SOURCE: Biochemical Journal, (15 Mar 2005) Vol. 386, No. 3, pp. 461-470.  
Refs: 42  
ISSN: 0264-6021 CODEN: BIJOAK  
COUNTRY: United Kingdom  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 029 Clinical Biochemistry  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 20050421  
Last Updated on STN: 20050421

L1 ANSWER 13 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN

TI Gene expression profiling of human erythroid progenitors by micro-serial analysis of gene expression.

AB We compared the expression profiles of highly purified human CD34 (+) cells and erythroid progenitor cells by micro-serial analysis of gene expression (microSAGE). Human CD34(+) cells were purified from granulocyte colony-stimulating factor-mobilized blood stem cells, and erythroid progenitors were obtained by cultivating these cells in the presence of stem cell factor, interleukin 3, and erythropoietin. Our 10,202 SAGE tags allowed us to identify 1354 different transcripts appearing more than once. Erythroid progenitor cells showed increased expression of LRBA, EEF1A1, HSPCA, PILRB, RANBP1, NACA, and **SMURF**. Overexpression of HSPCA was confirmed by real-time polymerase chain reaction analysis. MicroSAGE revealed an unexpected preferential expression of several genes in erythroid progenitor cells in addition to the known functional genes, including hemoglobins. Our results provide reference data for future studies of gene expression in various hematopoietic disorders, including myelodysplastic syndrome and leukemia.  
.COPYRGT. 2004 The Japanese Society of Hematology.

ACCESSION NUMBER: 2004455099 EMBASE  
TITLE: Gene expression profiling of human erythroid progenitors by micro-serial analysis of gene expression.  
AUTHOR: Fujishima N.; Hirokawa M.; Aiba N.; Ichikawa Y.; Fujishima M.; Komatsuda A.; Suzuki Y.; Kawabata Y.; Miura I.; Sawada K.-I.

CORPORATE SOURCE: Dr. M. Hirokawa, Department of Internal Medicine III, Akita University School of Medicine, 1-1-1 Hondo, Akita 010-8543, Japan. hirokawa@med.akita-u.ac.jp

SOURCE: International Journal of Hematology, (2004) Vol. 80, No. 3, pp. 239-245.  
Refs: 22  
ISSN: 0925-5710 CODEN: IJHEEY

COUNTRY: United States  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 022 Human Genetics  
025 Hematology  
029 Clinical Biochemistry  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 20041112  
Last Updated on STN: 20041112

L1 ANSWER 14 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN

TI Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.

AB **Smad** ubiquitin regulatory factor (**Smurf**) 1 binds to receptor-regulated Smads for bone morphogenetic proteins (BMPs) Smad1/5 and promotes their degradation. In addition, Smurf1 associates with transforming growth factor- $\beta$  type I receptor through the inhibitory **Smad** (I-**Smad**) Smad7 and induces their degradation. Herein, we examined whether Smurf1 negatively regulates BMP signaling together with the I-Smads Smad6/7. Smurf1 and Smad6 cooperatively induced secondary axes in *Xenopus* embryos. Using a BMP-responsive promoter-reporter construct in mammalian cells, we found that Smurf1

cooperated with I-Smad in inhibiting BMP signaling and that the inhibitory activity of Smurf1 was not necessarily correlated with its ability to bind to Smad1/5 directly. Smurf1 bound to BMP type I receptors via I-Smads and induced ubiquitination and degradation of these receptors. Moreover, Smurf1 associated with Smad1/5 indirectly through I-Smads and induced their ubiquitination and degradation. Smurf1 thus controls BMP signaling with and without I-Smads through multiple mechanisms.

ACCESSION NUMBER: 2003293267 EMBASE  
TITLE: Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.  
AUTHOR: Murakami G.; Watabe T.; Takaoka K.; Miyazono K.; Imamura T.  
CORPORATE SOURCE: K. Miyazono, Department of Biochemistry, Cancer Inst. Japan. Found. Cancer R., Tokyo 170-8455, Japan.  
miyazono-ind@umin.ac.jp  
SOURCE: Molecular Biology of the Cell, (1 Jul 2003) Vol. 14, No. 7, pp. 2809-2817.  
Refs: 29  
ISSN: 1059-1524 CODEN: MBCEEV  
COUNTRY: United States  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 029 Clinical Biochemistry  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 20030731  
Last Updated on STN: 20030731

L1 ANSWER 15 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN

TI Extracellular regulation of BMP signaling in vertebrates: A cocktail of modulators.

AB The transforming growth factor- $\beta$  (TGF- $\beta$ ) superfamily contains a variety of growth factors which all share common sequence elements and structural motifs. These proteins are known to exert a wide spectrum of biological responses on a large variety of cell types in both vertebrates and invertebrates. Many of them have important functions during embryonic development in pattern formation and tissue specification, and in adult tissues, they are involved in processes such as wound healing, bone repair, and bone remodeling. The family is divided into two general branches: the BMP/GDF and the TGF- $\beta$ /Activin/Nodal branches, whose members have diverse, often complementary effects. It is obvious that an orchestrated regulation of different actions of these proteins is necessary for proper functioning. The TGF- $\beta$  family members act by binding extracellularly to a complex of serine/threonine kinase receptors, which consequently activate Smad molecules by phosphorylation. These Smads translocate to the nucleus, where they modulate transcription of specific genes. Three levels by which this signaling pathway is regulated could be distinguished. First, a control mechanism exists in the intracellular space, where inhibitory Smads and Smurfs prevent further signaling and activation of target genes. Second, at the membrane site, the pseudoreceptor BAMBI/Nma is able to inhibit further signaling within the cells. Finally, a range of extracellular mediators are identified which modulate the functioning of members of the TGF- $\beta$  superfamily. Here, we review the insights in the extracellular regulation of members of the BMP subfamily of secreted growth factors with a major emphasis on vertebrate BMP modulation. .COPYRG. 2002 Elsevier Science (USA).

ACCESSION NUMBER: 2002378732 EMBASE  
TITLE: Extracellular regulation of BMP signaling in vertebrates: A cocktail of modulators.  
AUTHOR: Balemans W.; Van Hul W.  
CORPORATE SOURCE: W. Van Hul, Department of Medical Genetics, University of Antwerp, University Hospital, Antwerp 2610, Belgium.  
vhul@uia.ac.be  
SOURCE: Developmental Biology, (2002) Vol. 250, No. 2, pp. 231-250.  
Refs: 181  
ISSN: 0012-1606 CODEN: DEBIAO  
COUNTRY: United States  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 021 Developmental Biology and Teratology

LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 20021107  
 Last Updated on STN: 20021107

L1 ANSWER 16 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.  
 on STN

TI TGF- $\beta$  induces assembly of a Smad2-Smurf2 ubiquitin ligase complex that targets SnoN for degradation.

AB The receptor-regulated **Smad** proteins are essential intracellular mediators of signal transduction by the transforming growth factor- $\beta$  (TGF- $\beta$ ) superfamily of growth factors and are also important as regulators of gene transcription. Here we describe a new role for TGF- $\beta$ -regulated Smad2 and Smad3 as components of a ubiquitin ligase complex. We show that in the presence of TGF- $\beta$  signalling, Smad2 interacts through its proline-rich PPXY motif with the tryptophan-rich WW domains of Smurf2, a recently identified E3 ubiquitin ligases. TGF- $\beta$  also induces the association of Smurf2 with the transcriptional co-repressor SnoN and we show that Smad2 can function to mediate this interaction. This allows Smurf2 HECT domain to target SnoN for ubiquitin-mediated degradation by the proteasome. Thus, stimulation by TGF- $\beta$  can induce the assembly of a Smad2-Smurf2 ubiquitin ligase complex that functions to target substrates for degradation.

ACCESSION NUMBER: 2001211670 EMBASE

TITLE: TGF- $\beta$  induces assembly of a Smad2-Smurf2 ubiquitin ligase complex that targets SnoN for degradation.

AUTHOR: Bonni S.; Wang H.-R.; Causing C.G.; Kavsak P.; Stroschein S.L.; Luo K.; Wrana J.L.

CORPORATE SOURCE: J.L. Wrana, Program in Molecular Biology/Cancer, Samuel Lunenfeld Research Institute, Mount Sinai Hospital, 600 University Avenue, Toronto, Ont. M5G 1X5, Canada.  
 wrana@mshri.on.ca

SOURCE: Nature Cell Biology, (2001) Vol. 3, No. 6, pp. 587-595.  
 Refs: 39

ISSN: 1465-7392 CODEN: NCBIFN

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20010710

Last Updated on STN: 20010710

L1 ANSWER 17 OF 25 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Identifying protein-protein interactions, useful e.g. in drug development, comprises introducing into cells one or more prey proteins labeled with an epitope tag and one or more bait proteins labeled with a detectable substance.

AN 2004-315601 [29] WPIDS

AB WO2004023146 A UPAB: 20040505

NOVELTY - Identifying protein-protein interactions comprising prey proteins interacting with one or more bait comprises introducing one or more prey proteins in labeled with an epitope tag and one or more bait protein in cells labeled with a detectable substance.

DETAILED DESCRIPTION - Identifying protein-protein interactions comprising prey proteins interacting with one or more bait comprises:

(a) introducing one or more prey proteins in cells, where a prey is labeled with an epitope tag permitting separation of the prey protein from other proteins in the cells;

(b) introducing one or more bait protein in cells, where a bait protein is labeled with a detectable substance permitting detection of the bait protein and protein-protein interactions comprising a prey protein and the bait protein;

(c) inducing formation of protein-protein interactions between a prey and bait protein; and

(d) assaying for protein-protein interactions comprising a prey protein and bait protein by detecting the detectable substance.

INDEPENDENT CLAIMS are also included for:

- (1) quantitating protein-protein interactions;
- (2) determining an interactome for one or more bait protein;
- (3) determining the functions of gene product;
- (4) systematically and quantitatively analyzing protein-protein interactions in cell signaling;
- (5) determining the changes in an interactome of mitotic kinase during cell cycle progression;
- (6) analyzing protein-protein interactions in different cell types;
- (7) assaying for changes in protein-protein interactions in response to intracellular and extracellular factors;
- (8) identifying a potential modulator of signal transduction activity; and
- (9) an agent, modulator or inhibitor identified by a method of (8).

ACTIVITY - Antiinflammatory; Cytostatic.

No biological data given.

MECHANISM OF ACTION - None Given.

USE - The method and kits are useful in identifying, quantifying and analyzing protein-protein interactions. The method is useful in determining a disease or condition associated with a test protein, monitoring the course of therapy, conducting a drug discovery business and in detecting mutations in cellular proteins. The pharmaceutical composition is useful in treating and preventing a disease or condition associated with an abnormality in a signal transduction pathway, e.g. fibrosis, inflammation or cancer.

Dwg. 0/3

ACCESSION NUMBER: 2004-315601 [29] WPIDS

DOC. NO. NON-CPI: N2004-251489

DOC. NO. CPI: C2004-119632

TITLE: Identifying protein-protein interactions, useful e.g. in drug development, comprises introducing into cells one or more prey proteins labeled with an epitope tag and one or more bait proteins labeled with a detectable substance.

DERWENT CLASS: B04 D16 S03

INVENTOR(S): BARRIOS-RODILES, M; WRANA, J

PATENT ASSIGNEE(S): (MOUN) MOUNT SINAI HOSPITAL

COUNTRY COUNT: 105

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004023146	A2	20040318	(200429)*	EN	53
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS					
LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK					
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR					
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH					
PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC					
VN YU ZA ZM ZW					
AU 2003264211	A1	20040329	(200459)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004023146	A2	WO 2003-CA1354	20030905
AU 2003264211	A1	AU 2003-264211	20030905

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003264211	A1 Based on	WO 2004023146

PRIORITY APPLN. INFO: US 2002-408922P 20020906

L1 ANSWER 18 OF 25 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Novel isolated **Smurf** protein useful for inhibiting bone

AN  
AB

morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.

2001-071267 [08] WPIDS

WO 200077168 A UPAB: 20011129

NOVELTY - An isolated Smurf1 or Smurf2 protein (I), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) an isolated nucleic acid (II) encoding (I);
- (2) a vector (III) comprising (II);
- (3) a host cell (IV) comprising (III);
- (4) production of (I);
- (5) a transgenic non-human animal that expresses a human (I);
- (6) screening (M) for a modulator of **Smurf** activity, comprising detecting modulation of **Smurf** activity in the presence of a test compound relative to **Smurf** activity in the absence of the test compound;
- (7) an antibody (V) that specifically binds to (I);
- (8) an oligonucleotide or nucleic acid (VI) that specifically hybridizes to (II) under highly stringent conditions; and
- (9) promoting a bone morphogenic protein or transforming growth factor (TGF)- beta activation pathway in a cell, comprising suppressing expression of endogenous **Smurf** in the cell.

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - Negative regulator of **Smad** signal transduction; antagonist of BMP and TGF- beta signaling pathway.

The inhibition of Smad1 by Smurf1 was tested. By over expressing Smad1 and Smad2 together with various dosages of Smurf1 in Xenopus animal caps, the ability of Smurf1 to directly antagonize the mesoderm induction activities of Smad1 and Smad2, was tested. The results showed that expression of Smad1 alone induced ventral mesoderm, as demonstrated by expression of the ventral/posterior mesodermal markers Xhox3 and Xcad1. However, co-expression of Smurf1 and Smad1 blocked induction of these markers at all Smurf1 doses tested, demonstrating that Smurf1 can antagonize Smad1 activity.

USE - Expression of (I) from (III) in a cell is useful for inhibiting a bone morphogenic protein (BMP) or transforming growth factor- beta (TGF beta ) activation pathway in a cell (claimed). (I) is useful to block chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. (I) is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it by antagonizing or mimicking the activity of (I), respectively, and in screening assays for identifying specific ligands of (I). (I) is useful as an immunogen to generate antibodies that are useful to alter the BMP pathway by inhibiting (I) or for diagnostic purposes. (I) is useful for treating a disorder associated with BMP or TGF- beta activation, such as cancer. (I) or inhibitor of (I) can be delivered by a vector to modulate Smads, e.g. to prevent **Smurf** regulation of Smads where BMP or TGF beta activity is desired, such as in bone regeneration or to study **Smurf** regulator processes in vivo.

Dwg.0/18

ACCESSION NUMBER: 2001-071267 [08] WPIDS

DOC. NO. CPI: C2001-019969

TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.

DERWENT CLASS: B04 D16

INVENTOR(S): THOMSEN, G H; WRANA, J

PATENT ASSIGNEE(S): (HSCR-N) HSC RES & DEV LP; (UYNY) UNIV NEW YORK STATE RES FOUND

COUNTRY COUNT: 93

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
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 WO 2000077168 A2 20001221 (200108)\* EN 106  
 RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
 NL OA PT SD SE SL SZ TZ UG ZW  
 W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE  
 ES FI GB GD GE HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT  
 LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL  
 TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW  
 AU 2000056107 A 20010102 (200121)  
 EP 1192174 A2 20020403 (200230) EN  
 R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
 RO SE SI  
 JP 2003502064 W 20030121 (200308) 131  
 CN 1409722 A 20030409 (200345)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000077168	A2	WO 2000-US16250	20000612
AU 2000056107	A	AU 2000-56107	20000612
EP 1192174	A2	EP 2000-941398	20000612
		WO 2000-US16250	20000612
JP 2003502064	W	WO 2000-US16250	20000612
		JP 2001-504003	20000612
CN 1409722	A	CN 2000-811354	20000612

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000056107	A Based on	WO 2000077168
EP 1192174	A2 Based on	WO 2000077168
JP 2003502064	W Based on	WO 2000077168

PRIORITY APPLN. INFO: US 1999-138969P 19990611

L1 ANSWER 19 OF 25 JICST-EPlus COPYRIGHT 2005 JST on STN  
 TI Biomedicine research. 2. Water-soluble signal molecule. 2). Formation of the bone and transcriptional control.

AB Formation of the bone takes the process for the membranous ossification and the process for the enchondral ossification. In any process, transcription factors differentiating to the osteoblasts, which perform the osteogenesis in mesenchymal stem cells, seem to function. In this paper, transcription factors (Runx/Cbfa1, Osterix, ATF4, **Smad** and **Smurf**, TSH, ΔFosB) which bear the differentiation of the osteoblasts are outlined.

ACCESSION NUMBER: 1040891465 JICST-EPlus  
 TITLE: Biomedicine research. 2. Water-soluble signal molecule. 2). Formation of the bone and transcriptional control.  
 AUTHOR: NODA MASAKI; KONDO HISATAKA; USUI MICHIIHIKO; INOUE KEIICHI; NAKAJIMA KAZUHISA  
 CORPORATE SOURCE: Tokyo Medical and Dental Univ., Medical Res. Inst.  
 SOURCE: Idenshi Igaku Mook, (2004) no. 1, pp. 48-52. Journal Code: L3408B (Fig. 1, Ref. 10)  
 ISSN: 1349-2527  
 PUB. COUNTRY: Japan  
 DOCUMENT TYPE: Journal; Commentary  
 LANGUAGE: Japanese  
 STATUS: New

L1 ANSWER 20 OF 25 JICST-EPlus COPYRIGHT 2005 JST on STN  
 TI Ubiquitination and disease of the protein. Vital phenomenon and ubiquitination. Ubiquitination in the TGF-B signal transduction.  
 AB TGF - through serine/threonine kinase type receptor of the cell surface, B mainly activates intracellular transcription factor **Smad**, and it transmits the signal. For the adjustment of this **Smad** signal transduction, that the E3 ubiquitin kinase was related clarified

recently. In this paper, the signal transduction mechanism of TGF-B/BMP is simply introduced, and it combines with inhibited **Smad**, it actions negative feedback mechanism TGF-B superfamily 2 kinds of E3 ubiquitin kinase Arkadia. The action of 2 kinds of Smurf1 and Arkadia which adjusted the negative feedback mechanism of the TGF-B superfamily by combining with inhibited **Smad**, was outlined on TGF-B superfamily signal control mechanism by the ubiquitin proteasomes system. It is emphasized with inhibited **Smad**, and **Smurf** are concerned in the decomposition of receptor. In the meantime, though Arkadia is also similarly combined with **Smurf** in inhibited **Smad**, the decomposition of inhibited **Smad** is promoted, and the TGF-B/BMP signal is promoted.

ACCESSION NUMBER: 1040323261 JICST-EPlus  
TITLE: Ubiquitination and disease of the protein. Vital phenomenon and ubiquitination. Ubiquitination in the TGF-B signal transduction.  
AUTHOR: IMAMURA TAKESHI; TAJIMA YOSHITAKA; KOINUMA DAIZO  
CORPORATE SOURCE: Japanese Foundation for Cancer Res., Cancer Inst., JPN  
SOURCE: Gendai Iryo, (2004) vol. 36, no. 4, pp. 837-843. Journal Code: Z0273B (Fig. 3, Ref. 16)  
ISSN: 0533-7259  
PUB. COUNTRY: Japan  
DOCUMENT TYPE: Journal; Commentary  
LANGUAGE: Japanese  
STATUS: New

L1 ANSWER 21 OF 25 JICST-EPlus COPYRIGHT 2005 JST on STN  
TI Regulation of TGF-B signaling and its roles in progression of tumors.  
AB Transforming growth factor-B (TGF-B) is a potent growth inhibitor of most types of cells; therefore, perturbations of TGF-B signaling are believed to result in progression of various tumors. On the other hand, TGF-B has been shown to act as an oncogenic cytokine through induction of extracellular matrices, angiogenesis, and immune suppression. A wide variety of effects of TGF-B are mediated by physical interaction of signal transducer **Smad** proteins with various transcription factors. Among these. Runx3 plays a pivotal role in prevention of gastric cancer. TGF-B signaling is regulated by various mechanisms in the cytoplasm and nucleus. Inhibitory Smads (I-Smads) repress TGF-B signaling mainly by interacting with activated TGF-B receptors. **Smad** ubiquitin regulatory factors (Smurfs) play important roles in facilitating the inhibitory signals induced by I-Smads. In addition, the transcriptional co-repressors c-Ski and SnoN interact with Smads, and repress transcription induced by TGF-B. Abnormalities of these regulators of TGF-B signaling may thus participate in the progression of various tumors. (author abst.)

ACCESSION NUMBER: 1030234861 JICST-EPlus  
TITLE: Regulation of TGF-B signaling and its roles in progression of tumors.  
AUTHOR: MIYAZONO K; SUZUKI H  
IMAMURA T  
CORPORATE SOURCE: Univ. Tokyo, Tokyo  
Cancer Inst. Japanese Foundation For Cancer Res. (jfcr), Tokyo  
SOURCE: Cancer Sci, (2003) vol. 94, no. 3, pp. 230-234. Journal Code: F0633A (Fig. 3, Ref. 50)  
ISSN: 1347-9032  
PUB. COUNTRY: Japan  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English  
STATUS: New

L1 ANSWER 22 OF 25 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN  
TI Negative regulation of transforming growth factor-beta (TGF-beta) signaling by WW domain-containing protein 1 (WWP1).  
AB Smad7 negatively regulates transforming growth factor (TGF)-beta superfamily signaling by binding to activated type I receptors, thereby preventing the phosphorylation of receptor-regulated Smads (R-Smads), as

well as by recruiting HECT-type E3 ubiquitin ligases to degrade type I receptors through a ubiquitin-dependent mechanism. To elucidate the regulatory mechanisms of TGF-beta signaling, we searched for novel members of proteins that interact with Smad7 using a yeast two-hybrid system. One of the proteins identified was the WW domain-containing protein 1 (WWP1) that is structurally related to **Smad** ubiquitin regulatory factors (Smurfs), E3 ubiquitin ligases for Smads and TGF-beta superfamily receptors. Using a TGF-beta-responsive reporter in mammalian cells, we found that WWP1 inhibited transcriptional activities induced by TGF-beta. Similar to Smurfs, WWP1 associated with Smad7 and induced its nuclear export, and enhanced binding of Smad7 to TGF-beta type I receptor to cause ubiquitination and degradation of the receptor. Consistent with these results, WWP1 inhibited phosphorylation of Smad2 induced by TGF-beta. WWP1 thus negatively regulates TGF-beta signaling in cooperation with Smad7. However, unlike Smurfs, WWP1 failed to ubiquitinate R-Smads and SnoN. Importantly, WWP1 and Smurfs were expressed in distinct patterns in human tissues and carcinoma cell lines, suggesting unique pathophysiological roles of WWP1 and Smurfs.

ACCESSION NUMBER: 2004:461513 BIOSIS  
DOCUMENT NUMBER: PREV200400459747  
TITLE: Negative regulation of transforming growth factor-beta (TGF-beta) signaling by WW domain-containing protein 1 (WWP1).  
AUTHOR(S): Komuro, Akiyoshi; Imamura, Takeshi; Saitoh, Masao; Yoshida, Yoko; Yamori, Takao; Miyazono, Kohei; Miyazawa, Keiji [Reprint Author]  
CORPORATE SOURCE: Grad Sch MedDept Mol PatholBunkyo Ku, Univ Tokyo, 7-3-1 Hongo, Tokyo, 1130033, Japan  
keiji-miyazawa@umin.ac.jp  
SOURCE: Oncogene, (September 9 2004) Vol. 23, No. 41, pp. 6914-6923. print.  
ISSN: 0950-9232 (ISSN print).  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 1 Dec 2004  
Last Updated on STN: 1 Dec 2004

L1 ANSWER 23 OF 25 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.

AB **Smad** ubiquitin regulatory factor (**Smurf**) 1 binds to receptor-regulated Smads for bone morphogenetic proteins (BMPs) Smad1/5 and promotes their degradation. In addition, Smurf1 associates with transforming growth factor-beta type I receptor through the inhibitory **Smad** (I-**Smad**) Smad7 and induces their degradation. Herein, we examined whether Smurf1 negatively regulates BMP signaling together with the I-Smads Smad6/7. Smurf1 and Smad6 cooperatively induced secondary axes in *Xenopus* embryos. Using a BMP-responsive promoter-reporter construct in mammalian cells, we found that Smurf1 cooperated with I-**Smad** in inhibiting BMP signaling and that the inhibitory activity of Smurf1 was not necessarily correlated with its ability to bind to Smad1/5 directly. Smurf1 bound to BMP type I receptors via I-Smads and induced ubiquitination and degradation of these receptors. Moreover, Smurf1 associated with Smad1/5 indirectly through I-Smads and induced their ubiquitination and degradation. Smurf1 thus controls BMP signaling with and without I-Smads through multiple mechanisms.

ACCESSION NUMBER: 2003:356072 BIOSIS  
DOCUMENT NUMBER: PREV200300356072  
TITLE: Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.  
AUTHOR(S): Murakami, Gyo; Watabe, Tetsuro; Takaoka, Kunio; Miyazono, Kohei [Reprint Author]; Imamura, Takeshi  
CORPORATE SOURCE: Department of Biochemistry, The Cancer Institute of the Japanese Foundation for Cancer Research, Tokyo, 170-8455, Japan  
miyazono-ind@umin.ac.jp  
SOURCE: Molecular Biology of the Cell, (July 2003) Vol. 14, No. 7,

pp. 2809-2817. print.  
ISSN: 1059-1524 (ISSN print).  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 6 Aug 2003  
Last Updated on STN: 6 Aug 2003

L1 ANSWER 24 OF 25 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on  
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TI Specificity and complexity in **Smurf**-mediated **Smad**  
degradation.

ACCESSION NUMBER: 2002:133151 BIOSIS

DOCUMENT NUMBER: PREV200200133151

TITLE: Specificity and complexity in **Smurf**-mediated  
**Smad** degradation.

AUTHOR(S): Liang, Min [Reprint author]; Lin, Xia [Reprint author];  
Liang, Yao-Yun [Reprint author]; Feng, Xin-Hua [Reprint  
author]; DeBaKey, Michael E. [Reprint author]

CORPORATE SOURCE: Department of Surgery, Baylor College of Medicine, One  
Baylor Plaza, 139D, Houston, TX, 77030, USA

SOURCE: Molecular Biology of the Cell, (Nov, 2001) Vol. 12, No.  
Supplement, pp. 148a. print.  
Meeting Info.: 41st Annual Meeting of the American Society  
for Cell Biology. Washington DC, USA. December 08-12, 2001.  
American Society for Cell Biology.  
CODEN: MBCEEV. ISSN: 1059-1524.

DOCUMENT TYPE: Conference; (Meeting)  
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 6 Feb 2002  
Last Updated on STN: 26 Feb 2002

L1 ANSWER 25 OF 25 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on  
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TI Intracellular BMP signaling regulation in vertebrates: Pathway or  
network?.

AB Bone morphogenetic proteins (BMPs), members of the TGF-beta superfamily of  
secreted signaling molecules, have important functions in many biological  
contexts. They bind to specific serine/threonine kinase receptors, which  
transduce the signal to the nucleus through **Smad** proteins. The  
question of how BMPs can have such diverse effects while using the same  
canonical **Smad** pathway has recently come closer to an answer at  
the molecular level. Nuclear cofactors have been identified that  
cooperate with the Smads in regulating specific target genes depending on  
the cellular context. In addition, the pivotal role BMP signaling plays  
is underscored by the identification of factors that regulate members of  
this pathway at the cell surface, in the cytoplasm, and in the nucleus.  
Many of these factors are BMP-inducible and inhibit the BMP pathway, thus  
establishing negative feedback loops. Members of the BMP-**Smad**  
pathway can also physically interact with components of other signaling  
pathways to establish crosstalk. Finally, there is accumulating evidence  
that an alternative pathway involving MAP kinases can transduce BMP  
signals. The evidence and implications of these findings are discussed  
with an emphasis on early embryonic development of *Xenopus* and  
vertebrates.

ACCESSION NUMBER: 2001:540655 BIOSIS

DOCUMENT NUMBER: PREV200100540655

TITLE: Intracellular BMP signaling regulation in vertebrates:  
Pathway or network?.

AUTHOR(S): von Bubnoff, Andreas; Cho, Ken W. Y. [Reprint author]

CORPORATE SOURCE: Department of Developmental and Cell Biology, University of  
California, Irvine, CA, 92697-2300, USA  
kwcho@uci.edu

SOURCE: Developmental Biology, (November 1, 2001) Vol. 239, No. 1,  
pp. 1-14. print.  
CODEN: DEBIAO. ISSN: 0012-1606.

DOCUMENT TYPE: Article  
General Review; (Literature Review)

LANGUAGE: English  
ENTRY DATE: Entered STN: 21 Nov 2001  
Last Updated on STN: 25 Feb 2002

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## Search Results - Record(s) 1 through 4 of 4 returned.

### ☐ 1. Document ID: US 6727002 B2

L2: Entry 1 of 4

File: USPT

Apr 27, 2004

US-PAT-NO: 6727002

DOCUMENT-IDENTIFIER: US 6727002 B2

TITLE: EVOH and EVM in single- or multilayer products

DATE-ISSUED: April 27, 2004

#### INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hoch; Martin	Heinsberg			DE
Itter; Ulrich	Wuppertal			DE
Parg; Roland	Leverkusen			DE
Wrana; Claus	Cologne			DE
Schulte; Helmut	Krefeld			DE
Schwarz; Peter	Krefeld			DE
Ulrich; Ralph	Krefeld			DE

US-CL-CURRENT: [428/520](#); [264/173.19](#), [428/475.8](#), [428/476.3](#), [428/476.9](#), [428/522](#), [525/57](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KWIC	Draw Desc	Ima
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### ☐ 2. Document ID: US 6017755 A

L2: Entry 2 of 4

File: USPT

Jan 25, 2000

US-PAT-NO: 6017755

DOCUMENT-IDENTIFIER: US 6017755 A

TITLE: MADR2 tumour suppressor gene

DATE-ISSUED: January 25, 2000

#### INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Wrana; Jeffrey	Toronto			CA
Attisano; Liliana	Toronto			CA
Scherer; Stephen W.	Toronto			CA

US-CL-CURRENT: [435/320.1](#); [435/325](#), [536/23.5](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KWIC	Draw Desc	Ima
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### ☐ 3. Document ID: US 4679483 A

US-PAT-NO: 4679483

DOCUMENT-IDENTIFIER: US 4679483 A

TITLE: Dispenser and dispensing cassette

DATE-ISSUED: July 14, 1987

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Wrana; Josef B. V.	Sp.ang.nga			SE

US-CL-CURRENT: 89/1.51; 102/505, 244/137.4, 89/1.59

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KWIC	Draw Desc	Ima
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☐ 4. Document ID: US 4586439 A

L2: Entry 4 of 4

File: USPT

May 6, 1986

US-PAT-NO: 4586439

DOCUMENT-IDENTIFIER: US 4586439 A

TITLE: Cartridge for launching decoys

DATE-ISSUED: May 6, 1986

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Wrana; Josef B. V.	Sp.ang.nga			SE

US-CL-CURRENT: 102/438; 102/357, 102/505

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KWIC	Draw Desc	Ima
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### ☐ 1. Document ID: US 6775657 B1

L6: Entry 1 of 3

File: USPT

Aug 10, 2004

US-PAT-NO: 6775657

DOCUMENT-IDENTIFIER: US 6775657 B1

TITLE: Multilayered intrusion detection system and method

DATE-ISSUED: August 10, 2004

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Baker; Stephen M.	San Antonio	TX		

US-CL-CURRENT: 706/45; 706/50, 713/200, 713/201

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KWIC	Draw Desc	Ima
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### ☐ 2. Document ID: US 6687247 B1

L6: Entry 2 of 3

File: USPT

Feb 3, 2004

US-PAT-NO: 6687247

DOCUMENT-IDENTIFIER: US 6687247 B1

TITLE: Architecture for high speed class of service enabled linecard

DATE-ISSUED: February 3, 2004

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Wilford; Bruce	Los Altos	CA		
Dan; Yie-Fong	Cupertino	CA		

US-CL-CURRENT: 370/392; 370/412

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KWIC	Draw Desc	Ima
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### ☐ 3. Document ID: US 6684250 B2

L6: Entry 3 of 3

File: USPT

Jan 27, 2004

US-PAT-NO: 6684250

DOCUMENT-IDENTIFIER: US 6684250 B2

TITLE: Method and apparatus for estimating a geographic location of a networked entity

DATE-ISSUED: January 27, 2004

## INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Anderson; Mark	Westminster	CO		
Bansal; Ajay	Cupertino	CA		
Doctor; Brad	Broomfield	CO		
Hadjiyiannis; George	Boston	MA		
Herringshaw; Christopher	West Wardsboro	VT		
Karplus; Eli E.	Baden Wurttemberg			DE
Muniz; Derald	Midlothian	TX		

US-CL-CURRENT: 709/225; 370/392, 709/228

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KWIC	Draw Desc	Ima
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IBM Technical Disclosure Bulletins

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result set

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<u>L6</u>	L5 and (PPXY domain)	3	<u>L6</u>
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<u>L3</u>	L2 and l1	0	<u>L3</u>
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<u>L1</u>	thomsen.in.	677	<u>L1</u>

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